



Interferons in Viral Infections

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Message from the Guest Editors

Dear Colleagues,

The interferon (IFN) system represents the first line of defense against a wide range of viruses. Virus infection is sensed rapidly by a variety of cellular pattern recognition receptors in order to activate the intracellular signaling pathways. These signaling pathways activate the transcription factors, including the IFN regulatory factors (IRFs) and NF- κ B, to trigger the transcriptional induction of IFNs. IFNs, produced by the infected cells, are secreted and act on the infected and the yet uninfected cells to induce the IFN-stimulated genes (ISGs). The ISG-encoded protein products act as viral restriction factors by interfering directly with specific stages of the viral life-cycle. The virus-specific nature of ISGs has led to extensive research in the past decade in order to reveal new viral restriction mechanisms. In addition to functioning virus-specifically, some ISGs amplify the host IFN response and activate cell death pathways to further strengthen the antiviral state of the infected host.

This Special Issue is intended to highlight some of these new mechanisms of the IFN system that regulate viral replication and pathogenesis.





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Message from the Editor-in-Chief

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Electronic files or software regarding the full details of the calculation and experimental procedure, if unable to be published in a normal way, can be deposited as supplementary material.

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